Regulation of CVS (1)

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ILOs

By the end of this lecture the student should be able to:

1. Explain the arterial baroreceptor reflex
2. Explain its physiological significance
3. Describe the atrial stretch receptors reflex.
4. Compare the physiological significances of the above two reflexes

Regulation of heart rate (HR)

- The heart rate is generated by the Sinoatrial Node (SAN), the pacemaker of the cardiac muscle (intrinsic regulation of HR).
- In normal adults the heart rate at rest is about 60-90 beats per minute (average HR is about 70 beats / minute).
- The rate is significantly higher in children.
- Newborn resting HR may be 120 bpm.
- Trained athletes typically have very low HRs at rest (≈ 50-60 beats / minute) due to high vagal tone.
- During sleep, the heart rate decreases by 10 to 20 beats per minute.
- It may increase during emotional excitement, and during muscular exercise, it may increase to rates well above 150 beats per minute.
- An increase in heart rate (more than 90 beats / minute) is known as tachycardia and a decrease (less than 60 beats / minute) is known as bradycardia.
- Although the heart generates and maintains its own beat, the rate of contraction can be changed to adapt to different situations.
- The frequency of discharge of SAN (which determines the HR) is affected (regulated) by nervous, chemical and other factors (extrinsic regulation of HR).
- Nervous regulation of HR is by cardiovascular centers which control the sympathetic and parasympathetic discharge to heart.

Cardiac Vagal Tone

- There is continuous inhibitory effect exerted by the vagus nerves (tonic vagal discharge) on the heart at rest.
- It dominates over the sympathetic tone (tonic discharge in the cardiac sympathetic nerves) at the SAN reducing its rhythmicity from about 100 to 70 beats/min.

Medullary Cardiovascular Control Centers

- The cardiovascular control centers are located in the medulla of the brain.
- These centers receive information from different parts of the body and regulate the function of CVS through controlling sympathetic and parasympathetic discharge to CVS.

Medullary Sympathetic center	**Medullary Parasympathetic center**
Rostral ventrolateral medulla (RVLM) = Vasomotor Center (VMC)	**Nucleus ambiguus & dorsal motor nucleus of vagus (called previously cardioinhibitory center, CIC)**
• Its discharge reaches heart & blood vessels via sympathetic nerves • During rest continuous signals are passed from VMC to sympathetic vasoconstrictor nerve fibers over the entire body → V.C. • Stimulation of VMC produces – Arteriolar constriction → peripheral resistance → increase systemic blood pressure – Venoconstriction → increase venous return & cardiac output – Increase heart rate (positive chronotropic effect) – Increase force of contraction (positive inotropic effect)	• It sends inhibitory signals (parasympathetic impulses) to the heart via vagal nerve to decrease heart rate and heart contractility

• It receives inhibitory signals from nucleus of the tractus solitarius (NTS) of the medulla	• It receives excitatory signals from NTS

N.B.
The VMC & CIC act in reciprocal manner

The activity of medullary cardiovascular centers is modified by signals from:

I. Receptors located within the cardiovascular system

1. Baroreceptors
2. Atrial stretch receptors
3. Peripheral chemoreceptors

II. Receptors within the body but outside the cardiovascular system

1. Reflex response to pain
2. Reflexes from receptors in exercising skeletal muscle

III. Higher centers

1. The cerebral cortex, limbic system and hypothalamus
2. Respiratory centers
3. Central chemoreceptors (Cushing reflex)

These inputs normally enable the cardiovascular centers to regulate heart function precisely in response to the needs of the body.

I. Control by reflexes initiated from the cardiovascular system

1. Arterial baroreceptors and baroreceptor reflex

- Baroreceptors are mechanoreceptors located in the carotid sinus and the aortic arch, and are sensitive to stretch.
- Their function is to detect changes in arterial pressure.
- They are most important receptors involved in the moment-to-moment (acute) regulation of blood pressure
- Baroreceptors are sensitive to changes in both mean arterial pressure (within a range varing between about 60 and 160 mm Hg) and pulse pressure.

- Baroreceptors are more sensitive to pulsatile pressure than to constant pressure.
- The carotid sinus baroreceptors are located in the wall of internal carotid artery just above the bifurcation of the common carotid into external and internal carotid branches.
- Aortic arch baroreceptors are found in the wall of the arch of the aorta.
- The nerves from baroreceptors are called buffer nerves.
- The afferent nerve fibers from the carotid sinus form a branch of the glossopharyngeal nerve, called the carotid sinus nerve. The fibers from the aortic arch form a branch of the vagus nerve, called the aortic depressor nerve.
- Signals from the carotid and aortic baroreceptors are transmitted through these buffer nerves to the nucleus of tractus solitarius of the medulla which cause changes in the activity of medullary cardiovascular centers (increases or decreases in outflow from the sympathetic and parasympathetic nervous systems).
- Baroreceptors are tonically (constantly) active, producing a baseline frequency of action potentials in their sensory neurons.
- At normal blood pressure levels (about 100 mmHg mean pressure), a burst of action potentials is transmitted from the baroreceptor to stimulate the cardiac vagal neurons that decreases heart rate (vagal tone). At lower mean pressures, the firing is reduced while at higher prssures, the firing is increased.

The baroreceptor reflex

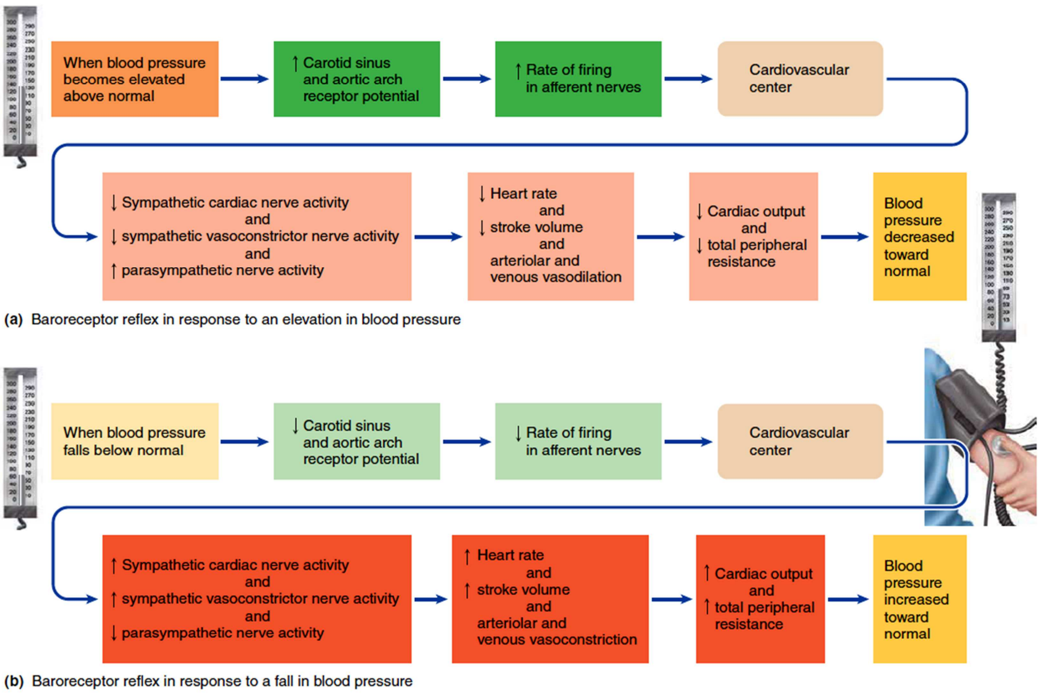
- Acute changes in arterial blood pressure reflexly elicit inverse changes in heart rate via the baroreceptors
- If for any reason mean arterial pressure rises above normal, the carotid sinus and aortic arch baroreceptors increase the rate of firing in their respective afferent neurons. On being informed by increased afferent firing that the blood pressure has become too high, the cardiovascular control center responds by decreasing sympathetic and increasing parasympathetic activity to the cardiovascular system.
- These efferent signals decrease heart rate, decrease stroke volume, and produce arteriolar and venous vasodilation, which in turn lead to a decrease in cardiac output and a decrease in total peripheral resistance, with a subsequent fall in blood pressure back toward normal.

Role of baroreceptors in short-term control of blood pressure:

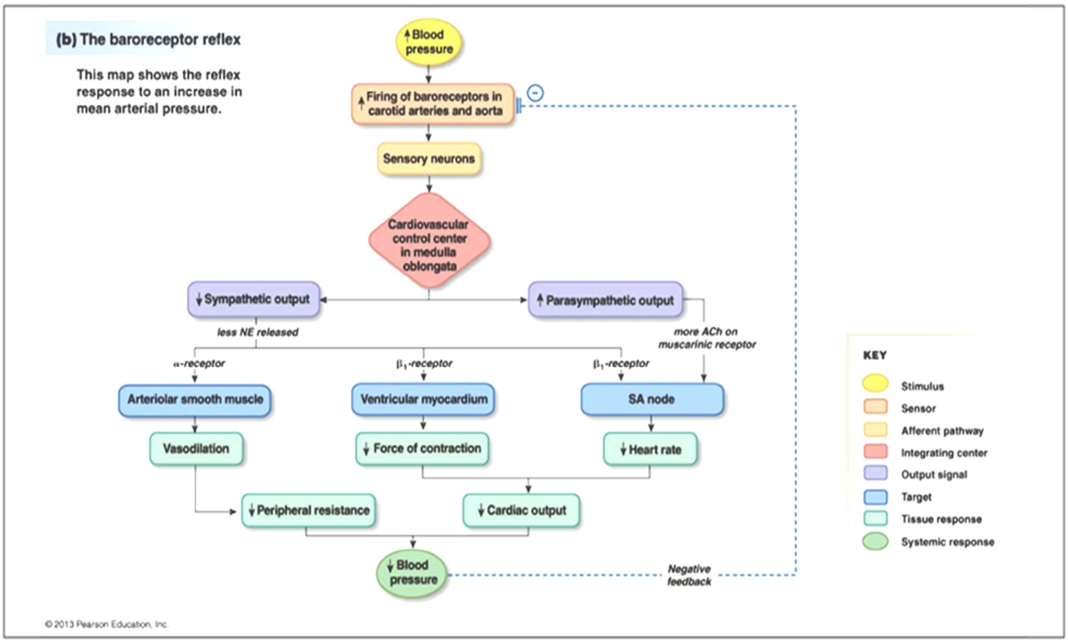
- Baroreceptors are very important in short-term control of arterial pressure.
- Baroreceptor reflex is responsible for the compensatory changes in pulse rate and blood pressure that occur in humans on standing up or lying down.
- Activation of the reflex allows for rapid adjustments in blood pressure in response to abrupt changes in posture, blood volume, cardiac output, or peripheral resistance.

Baroreceptor resetting

- In chronic hypertension, the baroreceptor reflex mechanism is "reset" to maintain an elevated rather than a normal blood pressure.



Baroreceptor reflexes to restore blood pressure to normal.



2. Atrial stretch receptors

There are two types of atrial receptors:

- Type A: discharges primarily during atrial systole.
- Type B: discharges during atrial diastole at the time of peak atrial filling.
  - Their rate of discharge increases when there is increase in the blood volume or the venous return.
  - These receptors are called low-pressure receptors.
  - They are pressure as well as volume controllers.

Atrial Reflex =Volume Reflex

- Distention of atrial stretch receptors type B by increased blood volume causes activation of atrial stretch receptors that result in:

1. Increased heart rate due to increased sympathetic nerve activity to SAN.
   - The cardiac response is highly selective and increase in sympathetic activity is restricted to the heart rate; there is no increase of sympathetic activity to peripheral arterioles or ventricular contractility.
2. Increase urine volume due to:
   - Inhibition of antidiuretic hormone (ADH) (vasopressin) release by the posterior pituitary gland which diminishes the reabsorption of water from renal tubules (main mechanism).
   - Inhibition of sympathetic vasoconstriction in afferent arterioles in the kidneys, leading to renal vasodilation (partial mechanism).

Both mechanisms reduce an increased blood volume back toward normal

3. Increased secretion of atrial natriuretic peptide (ANP) by the atria.
   - ANP has potent diuretic and natriuretic effects on the kidneys increasing urinary Na^+ and water excretion → increases urine volume → lowers blood volume.
   - ANP causes vasodilation (dilates resistance and capacitance blood vessels) → lowers blood pressure.
   - Thus, ANP is an important regulator of blood volume and blood pressure.